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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
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09/498,135 02/04/00 STONE

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020322
SNELL & WILMER
ONE ARIZONA CENTER
400 EAST VAN BUREN
PHOENIX AZ 85004-0001

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EXAMINER

ENEWOLD, J.

ART UNIT

PAPER NUMBER

1655

DATE MAILED:

05/31/00

Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary

Application No.

09/498,135

Applicant(s)

STONE, JOHN F.

Examiner

Jeanine A Enewold

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136 (a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133).

Status

- 1) ☒ Responsive to communication(s) filed on 04 February 2000.
- 2a) ☐ This action is **FINAL**. 2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 1-17 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 1-17 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claims _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☐ The drawing(s) filed on _____ is/are objected to by the Examiner.
- 11) ☐ The proposed drawing correction filed on _____ is: a) ☐ approved b) ☐ disapproved.
- 12) ☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

- 13) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).
- a) ☐ All b) ☐ Some * c) ☐ None of the CERTIFIED copies of the priority documents have been:
1. ☐ received.
2. ☐ received in Application No. (Series Code / Serial Number) _____.
3. ☐ received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).
- * See the attached detailed Office action for a list of the certified copies not received.
- 14) ☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. & 119(e).

Attachment(s)

- 15) ☒ Notice of References Cited (PTO-892)
- 16) ☒ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 17) ☒ Information Disclosure Statement(s) (PTO-1449) Paper No(s) 2.
- 18) ☐ Interview Summary (PTO-413) Paper No(s). _____.
- 19) ☐ Notice of Informal Patent Application (PTO-152)
- 20) ☐ Other: _____.

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DETAILED ACTION

Drawings

1. The drawings are objected by the draftsman (see PTO 948).

Claim Objections

2. Claims 16 and 17 are objected to because each of these claims recites "marking at lease some of the chromosome pieces". It is presumed that the recitation should read "marking at least some of the chromosome pieces.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

3. Claims 1-17 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method for facilitating diagnosis of Alzheimer's disease in women by exposing cells from the patient to a chromosome damaging agent and comparing the effect of the agent on the chromosome from the patient's cell with the effect on the chromosome from normal women wherein an increase in the amount of damage to the patient's chromosome as compared to normal chromosomes facilitates diagnosis of Alzheimer's disease, does not reasonably provide enablement for diagnosing any disease by exposing cells of a suspected diseased patient to any

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chromosome damaging agent, marking some of the chromosome fragments, and analyzing the fragments to determine whether cells were affected by the disease nor for diagnosing Alzheimer's disease by exposing cells of a suspected diseased patient to a chromosome damaging agent, marking some of the chromosome fragments, and analyzing the fragments to determine whether cells were affected by the disease. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to use the invention commensurate in scope with these claims.

The claims are broadly drawn to a method suitable for facilitating any disease diagnosis by exposing cells of a suspected diseased patient to any chromosome damaging agent, marking some of the chromosome fragments, and analyzing the fragments to determine whether cells were affected by the disease.

The specification teaches that chromosome analysis to determine diagnosis of disease may be performed by culturing cells and exposing the cells to a damaging agent. The exemplary embodiment of the invention is related to Alzheimer's disease. The specification fails to provide any working examples of the invention where specific Alzheimer patients and control patients were analyzed. The specification does not provide any population study, age of chromosomes, or individual range of stabilities.

The art teaches that gender differences exist in the study of genetic instability in Alzheimer's disease (AD). Cherry et al (Mutation Research, Vol. 275, pg. 57-67, 1992) teaches that breakage rates for bleomycin and methyl methane-sulfonate exposed cells were not significant in AD affected men when compared to controls. In contrast, AD

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women showed a significantly higher breakage rate for both drugs than those in age-matched control women (abstract). As seen in Figure 1, differences between male patients afflicted with AD and the old control patients can not be distinguished in either bleomycin or MMS breaks. Cherry states that "gender differences among normal age-matched controls may seriously confound the analysis of clastogen data within this AD population" (pg. 65, col. 1). Cherry continues to state that "in men, the drug sensitivity shown by the old controls completely obscures that seen in the AD group" (pg. 65, col. 1) thus indicating that bleomycin and MMS are not effective markers for AD. Thus, a gender difference appears to exist.

The art also teaches that not all diseases are sensitive to chromosome damaging agents. Robbins et al (J. of Neurology, Neurosurgery and Psychiatry, Vol. 48, pg. 916-923, 1985) teaches that lymphoblastoid lines from patients with amyotrophic lateral sclerosis are not hypersensitive to X-rays. Similarly, Parshad et al (PNAS, Vol. 93, pg. 5146-5150, 1996) teaches that three patients with amyotrophic lateral sclerosis, four patients with Parkinson disease and three patients with Huntington disease had normal results when subjected to fluorescent light tests. Thus, not all diseases are sensitive to chromosome damaging agents and would not be detected with the claimed method.

The art teaches that not all strand-braking agents induce chromosomal instability. Limoli et al (Cancer Research, Vol. 57, pg. 4048-4056, September 1997) studies five different agents (bleomycin, neocarzinostatin, hydrogen peroxide, restriction endonucleases, and ionizing radiation) to determine their capacity to induce delayed chromosomal instability (abstract). Restriction endonucleases and hydrogen peroxide

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however did not show any delayed chromosomal instability. Examination of cells treated with hydrogen peroxide showed no chromosome aberrations at the first mitosis. Further isolation of clones subjected to hydrogen peroxide treatment showed no delayed chromosomal instability. As seen in Table 4, very small percentages of aberrant metaphases were detected ranging from 0 to the largest at 12.5 (pg. 4052).

Thus based upon the teachings in the art and the lack of teachings in the specification, disease diagnosis based upon by exposing cells of a suspected diseased patient to a chromosome damaging agent, marking some of the chromosome fragments, and analyzing the fragments to determine whether cells were affected by the disease would be unpredictable. Since the art teaches that there are major gender differences between men and women and their response to chromosome damaging agents, disease diagnosis for both genders would be unpredictable based upon this method. It would be undue experimentation to determine how to distinguish AD affected men from normal old men in the control sample since there are no significant differences in breaks between the two populations when subjected to bleomycin, a chromosomal damaging agent. Further, since the art teaches several diseases which do not show any difference from normal controls when analyzed, disease diagnosis of any disease would be unpredictable. Additionally, not all diseases are linked to chromosomal instability for example those disease linked to pathogens, and microorganisms. Therefore, since the art has taught several diseases including amyotrophic lateral sclerosis, Parkinson disease and Huntington's disease which can not be distinguished from normal controls, disease diagnosis based upon exposing cells

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to a chromosomal damaging agent would be unpredictable. It would be undue experimentation for the skilled artisan to perform additional assays to determine which individuals are afflicted by the disease and which individuals are not. Finally, exposure to any chromosome damaging agent would not necessarily be predictable of genetic instability. As exemplified in Limoli, hydrogen peroxide, a strand-breaking agent, showed no significant chromosomal instability. Thus, based upon the lack of teaching in respect to gender differences, applicable disease, and applicable chromosome damaging agents, it would be unpredictable to practice the invention as broadly as claimed.

Applicant should note that amendment of the claims to the above enabled subject matter would introduce new matter.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

4. Claims 1-17 are rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention.

A) Claims 1-10 are indefinite because the claims do not recite a positive process step which clearly relates back to the preamble. The preamble states that the method suitable for facilitating disease diagnosis but the final process step is analyzing the marked chromosomal fragments to determine whether the cells were affected by

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disease. Therefore the claims are unclear as to whether the method is a method suitable for facilitating disease diagnosis or is analyzing the marked chromosomal fragments to determine whether the cells were affected by disease. There is no final process step which indicates how to determine whether the cells were affected by disease.

B) Claims 11-15 are indefinite because the claims do not recite a positive process step which clearly relates back to the preamble. The preamble states that the method is a method for analyzing an effect of disease on cells but the final process step is counting a number of marked chromosome pieces. Therefore the claims are unclear as to whether the method is a method for analyzing an effect of disease on cells or is a method for counting a number of marked chromosome pieces.

C) Claims 11-15 are indefinite over the recitation "a method of analyzing an effect of disease on cells". As written the method samples cells "suspected of being diseased" to determine "an effect of disease on cell". It is unclear how the method may determine "an effect of disease on cells" provided the cells are merely suspected to be diseased. If in fact the cells are not diseased, the effect of disease on cells would not be able to be determined. Further, it is unclear what "effect" is being studied. Thus, the metes and bounds of the claimed invention are unclear.

D) Claims 1-17 are indefinite over the recitation "marking at least some of the chromosomal fragments" because it is unclear whether the fragments are labeled, separated, or stained. Marking is not an art recognized term, thus the metes and bounds of the claimed invention are unclear.

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E) Claims 16 and 17 are indefinite because the claims do not recite a positive process step which clearly relates back to the preamble. The preamble states that the method is a method suitable for facilitating diagnosis of Alzheimer's disease but the final process step is measuring an amount of marked chromosome pieces (Claim 16) or comparing a number of marked chromosome pieces present in unaffected cells to sample cells. Therefore the claims are unclear as to whether the method is a method for facilitating diagnosis of Alzheimer's disease or measuring an amount of marked chromosome pieces (Claim 16) or comparing a number of marked chromosome pieces present in unaffected cells to sample cells (Claim 17).

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

5. Claims 1-4, 6-7, 11, 13-17 are rejected under 35 U.S.C. 103(a) as being unpatentable over Cherry et al. (Mutation Research, Vol. 275, pg. 57-67, 1992).

Cherry et al. (herein referred to as Cherry) teaches facilitating disease diagnosis by exposing cells of a suspected diseased patient to a chromosome damaging agent, marking some of the chromosome fragments, and analyzing the fragments to determine whether cells were affected by the disease. Specifically, peripheral blood lymphocytes from patients with Alzheimer's disease (AD) and controls were grown in culture for 72

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hours with phytohemagglutinin (mitogen)(pg. 60, col. 2)(limitations of Claims 2 and 3). Then the cells were treated with bleomycin, which causes an activated oxygen radical, or with methyl methane sulfonate (MMS) (chromosome damaging agents)(abstract)(limitations of Claim 6-7, 13, 15). Then cells were harvested, fixed on slides, and marked with Giemsa stain (limitations of Claim 4). 50 cells/ patient were scored for chromosome damage. Comparison between patients with Alzheimer's and control patients was performed to determine whether a significant difference existed (limitations of Claims 14, 16 and 17). As seen in Figure 1, bar charts are presented which show significant differences between AD women and control women with bleomycin (pg. 62). Cherry teaches that when considering women, bleomycin is a very effective marker for AD (pg. 65, col. 1).

Cherry does not explicitly teach a method of diagnosing Alzheimer's. However, Cherry does teach that significant differences in sensitivity to chromosome damaging agents as compared to control individuals.

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have modified the method of Cherry to predict the AD state of individuals. Based upon the teachings in Cherry that significant differences between AD women and control women was observed when cells were treated with bleomycin, the ordinary artisan would have been motivated to test unknown samples to determine the AD status of the individual. The ordinary artisan would expect that a significant portion of individual's cells afflicted with AD would show greater instability when treated with bleomycin. With the teachings of Cherry that significant

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differences between AD women and control women, the ordinary artisan would have recognized that the method of Cherry would be ideal to screen for AD. Thus there is a reasonable expectation of success to predicting the AD state of individuals based upon the teachings of Cherry.

6. Claims 1-2, 4, 11, 13-14, 16-17 are rejected under 35 U.S.C. 103(a) as being unpatentable over Chen et al (Mutation Research, Vol. 256, pg. 21-27, 1991).

Chen et al. (herein referred to as Chen) teaches facilitating disease diagnosis by exposing cells of a suspected diseased patient to a chromosome damaging agent, marking some of the chromosome fragments, and analyzing the fragments to determine whether cells were affected by the disease. Specifically, Chen teaches a sampling cells and transforming by Epstein-Barr virus to establish lymphoblastoid cell lines (pg. 22, col. 1). Cells were cultured in agar and subjected to irradiation (a chromosome damaging agent)(pg. 22, col. 2)(limitations of Claim 2). The colonies with 50 or more cells were isolated to determine the frequency of radiation-induced aberrations. The cells were fixed, spread on slide, and stained with Giemsa to mark the chromosomes (pg. 22, col. 2)(limitations of Claim 4). Upon studying of the cells, a higher frequency of chromosome-type lesions was observed in AD cells, indicating the cells from AD patients were more radiosensitive than normal patients (pg. 25, col. 1). 12 or 14 patients show sensitivities greater than cells from age-matched controls (pg. 26, col. 1).

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Chen does not explicitly teach a method of detecting Alzheimer's. However, Chen does teach significant differences in sensitivity to chromosome damaging agents as compared to control individuals.

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have modified the method of Chen to predict the AD state of individuals. Based upon the teachings in Chen that significant differences between AD and controls were observed when cells were treated with radiation, the ordinary artisan would have been motivated to test unknown samples to determine the AD status of the individual. The ordinary artisan would expect that a significant portion of individual's cells afflicted with AD would show greater instability when treated with radiation. With the teachings of Chen that 12 of 14 AD patient's cells exhibited significantly greater radiosensitivity than cells from controls, the ordinary artisan would have recognized that the method of Chen would be ideal to screen for AD. Thus there is a reasonable expectation of success to predicting the AD state of individuals based upon the teachings of Chen.

7. Claims 1-6, 11-17 are rejected under 35 U.S.C. 103(a) as being unpatentable over Parshad et al (PNAS, Vol. 93, pg. 5146-5150, May 1996).

Parshad et al. (herein referred to as Parshad) teaches a method for facilitating disease diagnosis by exposing cells of a suspected diseased patient to a chromosome damaging agent, marking some of the chromosome fragments, and analyzing the fragments to determine whether cells were affected by the disease. Specifically,

Parshad teaches sampling skin fibroblasts and blood from patients diagnosed with Alzheimer's and control patients. The heparinized blood was mixed with phytohemagglutini (mitogen) and incubated for 48 or 68 hours (limitations of Claims 2 and 3). The lymphocyte cultures were subjected to either fluorescent light or 254 nm UV light (chromosome damaging agent that causes free radical-induced DNA damage) (pg. 5147, col. 1, para. 3 and 4)(limitations of Claim 6). Moreover, the cells were then treated with beta-cytosine arabinoside (araC) or caffeine (repair retarding agents) (limitations of Claim 5 and 12). Chromatid breaks were quantitated using cytogenetic analysis of metaphase cells.

Prashad does not explicitly teach a method of detecting Alzheimer's. However, Prashad does suggest that the method described would be useful in predicting inheritance of familial AD and in supporting, or rendering unlikely, the diagnosis of sporadic AD in patients suspected of having the disease (abstract).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have modified the method of Prashad to predict the AD state of individuals. Based upon the teachings in Prashad that significant differences between AD and controls were observed when cells were treated with radiation and caffeine (pg. 5147, col. 2), the ordinary artisan would have been motivated to test unknown samples to determine the AD status of the individual. The ordinary artisan would expect that a significant portion of individual's cells afflicted with AD would show greater instability when treated with radiation. With the teachings of Prashad that 5 sporadic AD and three DS donors had elevated chromosomal breakage than cells

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from controls (pg. 5149, Table 6), the ordinary artisan would have recognized that the method of Prashad would be ideal to screen for AD. Thus there is a reasonable expectation of success to predicting the AD state of individuals based upon the teachings of Prashad.

8. Claims 1-2, 6-11, 13-15 are rejected under 35 U.S.C. 103(a) as being unpatentable over Gorczyca et al (Cancer Research, Vol. 53, pg. 1945-1951, April 1993).

Gorczyca et al. (herein referred to as Gorczyca) teaches a method of detecting DNA strand breaks by in situ terminal deoxynucleotidyl transferase and nick translation. Gorczyca teaches sampling peripheral blood cells from patients with CML and culturing the cells (pg. 1945-1946)(limitations of Claim 2). The samples were cultured with DNA topoisomerase I (CAM) and topoisomerase II inhibitors (TN) (chromosomal damaging agents) and fixed (pg. 1946, col. 1). Further, the cells were subjected to in situ assays including the NT and TdT assay. For the NT assay, the cells were suspended with nick translation buffer, dATP, dGTP and dCTP and biotin-16-dUTP (pg. 1946, col. 1), the incubated with fluoresceinated avidin (limitations of Claim 8 and 9). For the TdT assay, fixed cells were suspended in a solution containing biotin-16-dUTP and dATP, dGTP and dCTP, this incubated with fluorescented avidin (limitations of Claims 8 and 9). Gorczyca teaches that the advantages of TdT or NT assays include the direct labeling of 3'-OH termini of the DNA breaks (pg. 1950, col. 2)(Limitation of Claim 6). Further, image analysis or flow cytometry was performed to detect fluorescence emissions from

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each cell and the data was stored and analyzed (pg. 1946, col. 1)(limitations of Claim 10). As seen in Figure 7 different patterns existed between control and DNA topoisomerase inhibitors. Furthermore, Figure 9 illustrates a before treatment and during treatment monitoring of the disease.

Gorczyca does not explicitly teach a method of disease diagnosis. However, Gorczyca does suggest that the method described would be useful for applications to clinical material to study the induction of apoptosis in tumors during treatments and as a prognostic marker.

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have modified the method of Gorczyca to predict the disease state of individuals. Based upon the teachings in Gorczyca that differences between controls and CML cells were observed when cells were treated with chromosome damaging agents, the ordinary artisan would have been motivated to test samples to determine the disease status of the individual. Gorczyca's suggestion that the method described would be useful for applications to clinical material to study the induction of apoptosis in tumors during treatments and as a prognostic marker provide a reasonable expectation of success to predicting the disease state of individuals.

9. Claims 8-10 are rejected under 35 U.S.C. 103(a) as being unpatentable over Cherry et al. (Mutation Research, Vol. 275, pg. 57-67, 1992) as applied to Claims 1-4,

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6-7, 11, 13-17 above, and further in view of Gorczyca et al (Cancer Research, Vol. 53, pg. 1945-1951, April 1993).

This rejection is applied to a narrower embodiment where Alzheimer's is detected using terminal deoxynucleotidyl transferase and nick translation assays.

Cherry does not specifically teach labeling the chromosome fragments with dNTP and exposing the fragments to a fluoresceinated material.

However, Gorczyca et al. (herein referred to as Gorczyca) teaches a method of detecting DNA strand breaks by in situ terminal deoxynucleotidyl transferase and nick translation. Gorczyca teaches sampling peripheral blood cells and culturing the cells (pg. 1945-1946)(limitations of Claim 2). After treatment the cells were subjected to in situ assays including the NT and TdT assay. For the NT assay, the cells were suspended with nick translation buffer, dATp, dGtp and dCTP and biotin-16-dUTP (pg. 1946, col. 1), the incubated with fluoresceinated avidin (limitations of Claim 8 and 9). For the TdT assay, fixed cells were suspended in a solution containing biotin-16-dUTP and dATP, dGTP and dCTP, this incubated with fluorescented avidin (limitations of Claims 8 and 9). Gorczyca teaches that the advantages of TdT or NT assays include the direct labeling of 3'-OH termini of the DNA breaks (pg. 1950, col. 2)(Limitation of Claim 6). Further, image analysis or flow cytometry was performed to detect fluorescence emissions from each cell and the data was stored and analyzed (pg. 1946, col. 1)(limitations of Claim 10).

Therefore, it would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have modified the method of Cherry to include

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the labeling of the chromosomal fragments with biotin-16-dUTP and exposing to fluoresceinated avidin as taught by Gorczyca. The ordinary artisan would be motivated to have performed the method of Cherry and labeled the fragments with biotin and fluoresceinated in order to allow rapid detection with flow cytometry and amenable to automation as taught by Gorczyca.

Conclusion

10. No claims allowable over the art.


11. The prior art made of record and not relied upon is considered pertinent to applicant's disclosure.

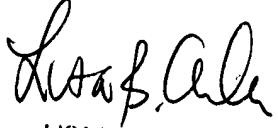
A) Prashad et al (JAGS, Vol. 46, No. 10, pg. 1331-1333, October 1998)- teaches exposing cells to DNA damaging agents, DNA repair inhibitors and quantitating breaks.

12. Any inquiry concerning this communication or earlier communications from the examiner should be directed to examiner Jeanine Enewold whose telephone number is (703) 306-5817. The examiner can normally be reached Monday-Thursday from 7:00AM to 4:30 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Jones, can be reached on (703) 308-1152. The fax number for this Group is (703) 305- 3014.

Any inquiry of a general nature should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Jeanine Enewold
May 24, 2000 


LISA B. ARTHUR
PRIMARY EXAMINER
GROUP 1600 1600